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Case Report

Headache and high ESR: A cautionary tale

V Garikipati, S Nair & M Prevett

Key Learning Points

- New persistent headache and raised ESR in a patient aged over 50 years should always suggest the possibility of temporal arteritis; however if symptoms do not improve within 72 hours of starting steroids, alternative causes must be considered.
- In the absence of raised intracranial pressure, a painful 6th nerve palsy in adults is most frequently caused due to neoplastic or inflammatory causes.
- Intracranial complications of sinusitis are associated with significant mortality; prompt diagnosis and treatment can result in complete recovery.

Abstract

A 69-year-old woman with new persistent right temporal headache and high ESR was diagnosed with temporal arteritis in primary care. She was started on steroids, but developed a right 6th nerve palsy with continuing headache and raised inflammatory markers. Investigation revealed a sphenoid sinus abscess with intracranial extension and extensive cerebral venous sinus thrombosis. The abscess was drained endoscopically and she was treated with antibiotics and anticoagulation. Over the course of two months she made a complete recovery.

Keywords

Temporal arteritis, sinusitis, cerebral venous sinus thrombosis.

Dr Venkat Garikipati
MRCP(UK)
Registrar Neurology
Wessex Neurological Centre

Dr Martin Prevett
DM, FRCP (UK)
Consultant Neurologist

Mr Salil Nair
FRCS
Consultant Skull Base Surgeon and Rhinologist

Correspondence:
Dr Venkat Garikipati
Specialist Registrar in Neurology
Mailpoint 101,
Wessex Neurological Centre
Southampton General Hospital
Tremona Road
Southampton
SO16 6YD
Email: garisat@gmail.com

Case History

A 69-year-old woman was referred to hospital with horizontal diplopia and a right sided temporal headache. She had been unwell for two weeks with intermittent fever, sweats and anorexia. The headache had been present for five days and an ESR measured three days before admission was 61. Temporal arteritis was suspected and she was started on Prednisolone 40 mgs daily. The day before admission she developed horizontal diplopia worse on gaze to the right. By the time of admission, there had been slight improvement in the headache, but she still had significant pain requiring opiate analgesics. The pain was intense and throbbing. It was worse at night and was exacerbated by touch. The pain increased with jaw opening, but there was no jaw claudication.

She had a history of diabetes (diet controlled) , hypertension, lipodystrophy, chronic sinusitis and asthma. She was treated with Omeprazole, Bendroflumethiazide, Candesartan, Raloxefene, Fluticasone nasal spray, Beclomethasone inhaler and Salbutamol inhaler.

On examination she was afebrile and there was no meningism. The fundi were normal. There was a right sixth nerve palsy. There was tenderness in the right temporal area, but the superficial temporal arteries were not thickened. The remainder of the neurological examination and systemic examination were unremarkable.

On admission, ESR was 86 (1-20) and CRP was 65 (0-7.5). CT brain showed opacification of the sphenoid sinus (Figure 1a) with loss of continuity of its bony margins (Figure 1b). Magnetic Resonance Imaging showed the sphenoid sinus to be filled with fluid, which had a restricted pattern on diffusion weighted images suggestive of pus. In the region of the bone erosion seen on CT there was enhancement extending through to the dural surface along the posterior aspect of the clivus and into the region of Meckel's cave (Figure 2). Asymmetry of the sigmoid sinuses was noted and a subsequent CT venogram showed thrombosis of the right cavernous sinus with extension into the right sigmoid sinus and internal jugular vein (Figure 3). The prednisolone was discontinued. She was commenced on intravenous Cefotaxime, Metronidazole and Rifampicin and an ENT opinion was sought. Nasal endoscopy showed mucosal oedema with a purulent discharge from both sphenoid ostia. Bilateral endoscopic sphenoidotomies were performed with drainage of pus which grew *streptococcus intermedius*. Following the procedure she was anticoagulated with low molecular weight heparin.

The 6th nerve palsy resolved within three days, but she continued to experience an intermittent fever for one week following drainage of the sinuses. Thirteen days after starting treatment she developed a right-sided sensorineural deafness. A repeat MRI brain showed no new changes to explain this. After two weeks of intravenous antibiotics she was

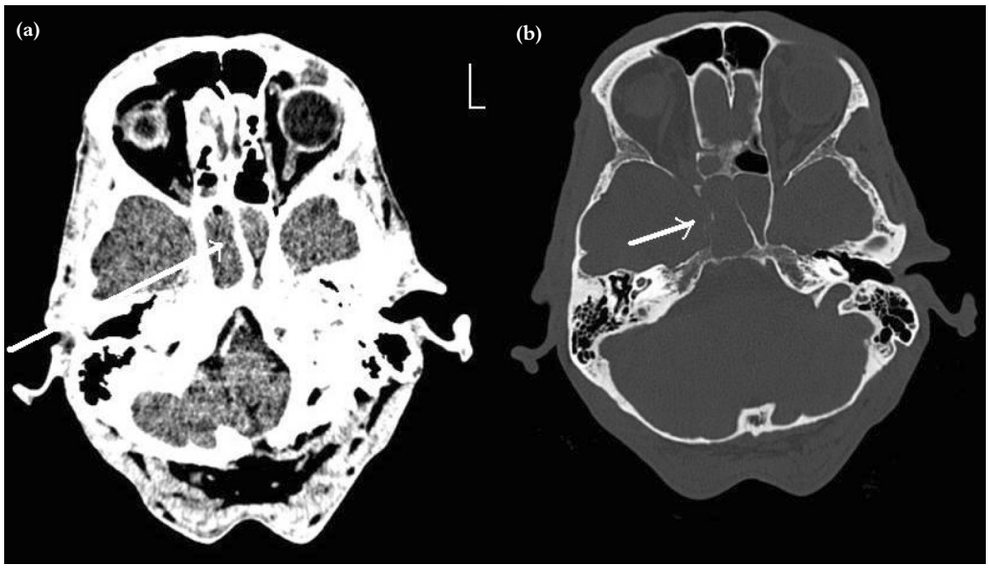

Figure 1. (a) Opacification of the sphenoid sinus. (b) Bony windows showing breakdown of the walls of the right sided sphenoidal walls.

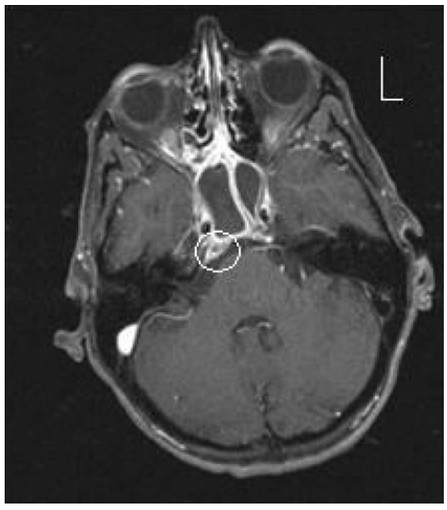

Figure 2. Extension of the inflammation to the clivus on contrast enhanced MRI.

discharged on a 6 week course of oral Coamoxiclav 375mgs tds and Amoxicillin 750mgs tds, and the low molecular weight heparin was switched to Warfarin.

Following discharge from hospital she made good progress with gradual resolution of the headache and improvement of the inflammatory markers (Table 1).

The right sided hearing loss also improved. Follow up MRI two months later showed residual mucosal thickening and opacification within the sphenoid sinus , and persistent occlusion of the thrombosed venous sinuses. There were no signs of raised intracranial pressure, and the warfarin was continued for 6 months.

Discussion

New onset headache and raised ESR in a patient over 50 years of age should always suggest the possibility of temporal arteritis, but as this case illustrates it is important to adhere to diagnostic criteria[1] (Table 2) and consider alternative diagnoses if the criteria are not satisfied.

Although our patient had a focal temporal headache and tenderness with raised inflammatory markers, there was no thickening of the superficial temporal artery and significant headache was still present 3 days after steroid treatment had been started. In addition, although cranial nerve palsies, fever and anorexia may all occur with temporal arteritis, the sixth nerve palsy occurred after starting steroids. These features prompted further investigation and an eventual diagnosis of a sphenoid sinus

Bloods	4 days before admission	On arrival	Preop, antibiotics started	4 days post op	2 wks post op	4 wks post op	10 wks post op	14 wks post op
CRP/mg/L (0-7.5)		65	221	66	21	5	7	6
ESR/mm 1st hr(1-20)	61	86	60	94		43	34	23

Table 1. Inflammatory markers.

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A.	Any new and persistant headache fulfilling criteria C and D
B.	At least one of the following:
	1. Swollen tender scalp artery with elevated erythrocyte sedimentation rate (ESR) and/or C reactive protein (CRP)
	2. temporal artery biopsy demonstrating giant cell arteritis
C.	Headache develops in close temporal relation to other symptoms and signs of giant cell arteritis
D.	Headache resolves or greatly improves within 3 days of high-dose steroid treatment

Table 2. Diagnostic criteria for temporal arteritis.

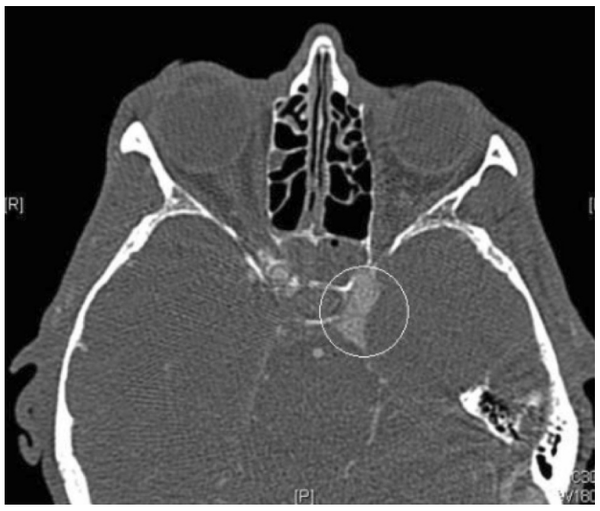

Figure 3. CT venogram showing absence of contrast in the right cavernous sinus due to thrombosis. Contrast is seen in the normal left cavernous sinus (circle).

abscess with intracranial extension complicated by cerebral venous sinus thrombosis.

In patients with diabetes and hypertension, infarction of the nerve is a common cause of an isolated 6th nerve palsy. However when there is a significant headache , neoplastic or inflammatory disorders are more likely. A sixth nerve palsy may also be a feature of raised intracranial pressure (the 'false localising' sign).

Suppurative complications of sinusitis are uncommon, but they are a potential cause of mortality and morbidity. Complications include osteomyelitis and sub-periosteal abscess. Infection in the frontal, ethmoid and sphenoid sinuses may extend intracranially resulting in meningitis or abscess formation. The carotid artery and cavernous sinuses are intimately related to the lateral wall of the sphenoid sinus. Infections that involve either of these structures may lead to development of aneurysms or thrombus. Infection of any of the sinuses may spread to the orbit causing orbital cellulitis and ,if advanced , cavernous sinus thrombosis.

The estimated incidence of intracranial complications of sinusitis varies considerably between studies, ranging from 0.5% to 24% of patients hospitalized with sinusitis. This figure appears to have declined considerably in recent decades with improvements in the diagnosis and treatment of sinusitis. [2,3,4] Studies have shown that 45% of patients with intracranial complications of sinusitis may also have an orbital cellulitis or frontal swelling.[5]

Diagnosis and management are challenging and require close cooperation between otolaryngology, neurosurgery, microbiology and neurology. In a large study of 219 patients with intracranial complications of sinusitis, 22 patients had meningitis, 127 subdural empyema, 38 brain abscess, 15 combined brain abscess and subdural empyema and 17 extradural empyema.[3] The most frequent presenting signs were fever (68%) and headache (54%). The most common localizing neurological sign was hemiparesis (35.5%). Orbital inflammation was present in 41.5% of patients. Despite advances in treatment and availability of imaging for early and accurate diagnosis, the mortality from intracranial complications of sinusitis remained significant. There were 35 deaths (16%). The highest mortality rate was recorded in patients with meningitis (45%) followed by brain abscess (19 %) and subdural empyema (11%).

Although intracranial complications of sinusitis have declined in incidence, they should not be forgotten in patients presenting with focal headache and raised inflammatory markers. Prompt diagnosis and treatment before the development of meningitis, empyema or cerebral abscess will increase the chance of full recovery.

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